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Individual fitness and phenotypic selection in age-structured populations with constant growth rates

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Abstract. Powerful multiple regression-based approaches are commonly used to measure the strength of phenotypic selection, which is the statistical association between individual fitness and trait values. Age structure and overlapping generations complicate determinations of individual fitness, contributing to the popularity of alternative methods for measuring natural selection that do not depend upon such measures. The application of regression-based techniques for measuring selection in these situations requires a demographically appropriate, conceptually sound, and observable measure of individual fitness. It has been suggested that Fisher's reproductive value applied to an individual at its birth is such a definition. Here I offer support for this assertion by showing that multiple regression applied to this measure and vital rates (age-specific survival and fertility rates) yields the same selection gradients for vital rates as those inferred from Hamilton's classical results. I discuss how multiple regressions, applied to individual reproductive value at birth, can be used efficiently to estimate measures of phenotypic selection that are problematic for sensitivity analyses. These include nonlinear selection, components of the opportunity for selection, and multilevel selection.

Key words: age structure; aging; evolution; fitness; multilevel opportunity; population demography; reproductive value; selection; senescence.

INTRODUCTION

The study of phenotypic evolution is concerned with describing how natural selection affects evolutionary changes in trait values across generations. Guided largely by quantitative genetic principles, this descriptive framework partitions phenotypic change into two necessary and sufficient components. First, phenotypic selection describes how individuals in one generation (the ancestral population) are represented in the next (the descendent population). Relative fitness is the weighting of this representation that is attributed to each member of the ancestral population. Second, inheritance is the fidelity with which ancestral phenotypes are transmitted to descendants. Genes are usually regarded as the primary mechanism of inheritance. A central concept of phenotypic selection methodologies is the notion that fitness is a quantifiable attribute of individuals. Estimates of phenotypic selection are often made using multiple regressions, where the relative fitness of individuals is the dependent variable, and a suite of traits that describe values pertaining to individuals are the independent variables (Lande and Arnold 1983, Phillips and Arnold 1989, Brodie et al. 1995).

Because inheritance is defined by associations between two generations (Falconer and Mackay 1996, Lynch and Walsh 1998), phenotypic selection is sensibly quantified on the scale of generations. This presents a problem with some forms of age-structure, however, as the notion of a generation is obscured when individuals of different ages reproduce with one another. One solution that has been adopted by life history theory has been to dispense with the notion of individual fitness altogether. Instead, growth rates are associated with populations, and "sensitivities" measure the strength of associations between hypothetical perturbations in these growth rates and the population mean of some character of interest. These sensitivities are interpreted as selection differentials (Lande 1982, Charlesworth 1994). Applied to vital rates, or age-specific survival and reproduction, these quantify the force of natural selection acting upon these rates. Two methods have been employed to define vital rate sensitivities: implicit differentiation (Hamilton 1966) and matrix population analysis (Caswell 1978). These methods yield the same results (see Eqs. A.7 and A.10 in the Appendix).

Because growth rates are properties of groups, they cannot describe individuals. This is not a problem for selection estimation procedures that work by analyzing differences among collections of individuals that are grouped by common vital rates (Hamilton 1966, Caswell 1978, Caswell 2001), genotypes (Charlesworth and Charlesworth 1973, Charlesworth 1994), or phenotypes (Lande 1982). However, methods of selection estimation

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that apply the concept of growth rates to individuals (McGraw and Caswell 1996) are problematic as statistical bias leads to the situation in which the mean growth rate taken over all individuals in a population can differ greatly from the population growth rate (Lenski and Service 1982). This precludes the general implementation of multiple regression methodologies and has left a methodological void in the study of phenotypic selection in age-structured populations. This deficiency is illustrated by three examples of evolutionarily relevant measures that are either hindered or entirely prevented without valid measurements of individual fitness. First, the among-individual variation in relative fitness (the opportunity for selection) is a fundamentally important measure of the capacity of a population to evolve greater fitness through natural selection (Crow 1958, O'Donald 1970, Arnold and Wade 1984, Hersch and Phillips 2004, Moorad and Wade 2013). Second, contextual analysis is a multiple regression-based method for quantifying causes of social evolution by partitioning multilevel trait selection into components of individual-level selection and group-level selection (Heisler and Damuth 1987, Goodnight et al. 1992, Okasha 2004). Third, nonlinear selection for phenotypes, including stabilizing, diversifying, and correlational selection, is defined in terms of multiple regressions of individual fitness measures upon linear and higher-order combinations of phenotypes (Lande and Arnold 1983, Phillips and Arnold 1989, Brodie et al. 1995, Rice 2004). These applications require a definition of fitness that is attributable to individuals, directly observable, and accounts for overlapping generations and population growth.

Some studies have asserted that the fitness of an individual is equal to its reproductive value at birth (Peters and Keightley 2000, Moorad 2013a, b). Recently, I supported this claim by estimating selection gradients for age-specific survival and fertility in a human population in two ways (Moorad 2013a). The first way was to find the conventional "sensitivities" obtained through implicit differentiation (Hamilton 1966, Charlesworth 1994). The second way was to apply multiple regressions using individual reproductive value at birth as the dependent variable and the individuals' age-specific survival and fertility as independent variables. Both methods yielded identical estimates of selection gradients for vital rates that demonstrated variation (several hundred cohort-specific vital rates were analyzed), and I interpreted this agreement as support for the assertion that individual fitness and reproductive value at birth were equivalent. I argued that Fisher's verbal definition of reproductive value (Fisher 1958) applied to individuals at birth and George Price's definition of fitness (Price 1970, 1972, Frank 1995) were harmonious, and I noted that relative lifetime reproductive output and reproductive value converged in static populations ($r = 0$), but I offered no

mathematical proof for the equivalency of relative fitness and individual reproductive value.

Here, I offer further evidence for this equivalency by re-deriving Hamilton's sensitivities (Hamilton 1966) using only the reproductive values of individuals at birth and the covariance definition of selection (Robertson 1966, Price 1970, 1972) applied to multiple characters (Lande and Arnold 1983). In this way, I provide confirmation of Hamilton's results from a conventional individual-based multivariate phenotypic selection perspective. More importantly, the definition of individual fitness used here links powerful regression-based analytical tools to issues relating to life history evolution in populations with overlapping generations. I illustrate this point by explaining how individual fitness in age-structured populations can be used to study selection in ways that were unavailable to previous methods. I also discuss the capacity for the multiple regression framework to allow more complicated fitness functions to be used to make more nuanced measures of selection for vital rates, such as may arise with sexually dimorphic vital rates.

METHODS

The assertion.—Fisher's reproductive value (1958), v_{0i} , of any individual i taken at birth is the fitness of that individual, w_i

$$w_i = v_{0i} = \sum_{y=0}^{\infty} \lambda^{-y} l_{yi} m_{yi} \quad (1)$$

where l_{yi} and m_{yi} are individual values of cumulative survival (constrained to be zero or one) and age-specific reproduction at some age y , respectively. The intrinsic rate of population growth is λ , which is equal to the exponential of the Malthusian rate of population increase r . It is assumed here that this rate is constant over time. As explained by classical life-history theory (Fisher 1958, Hamilton 1966, Charlesworth 1994), this rate is solved using the Euler-Lotka equation

$$\sum_{y=0}^{\infty} \lambda^{-y} \bar{l}_y \bar{m}_y = 1 \quad (2)$$

where at some age y , \bar{l}_y is the cumulative survival rate of the population, and \bar{m}_y is the mean reproductive output of the surviving fraction of the population. Note the additive relationship between vital rates and reproductive value demonstrated in Eq. 1 ensures that the mean of reproductive values is the same as the population-level reproductive values (Eq. 2). Also note that the mean reproductive value of individuals at birth taken over the population is one (Fisher 1958, Crow 2002). If the assertion that v_0 is fitness is true, then it must follow that v_0 is also relative fitness. Classical phenotypic selection theory that neglects overlapping generations (Lande and Arnold 1983, Arnold and Wade 1984) converts individual absolute fitness (or total lifetime reproduction) into relative fitness by dividing by the

population mean. The Euler-Lotka equation serves the same purpose in the more general case where generations are allowed to overlap by accounting for population growth and the reproductive timing of individuals.

A simple algebraic proof presented in the Appendix demonstrates that multiple regression of individual reproductive values at birth (Eq. 1) upon individual vital rate values yields selection gradients that are equivalent to the vital rate sensitivities obtained by Hamilton (1966), who defined fitness using the Malthusian parameter r . The two results differ only in the scaling of time over which fitness is accounted: the individual-fitness method defines phenotypic selection on the scale of generations and the sensitivity-based approach describes selection on the scale of arbitrarily defined time units. As generation time is easily determined in terms of time units (see Appendix: Eq. A.7), sensitivities also can be used to describe vital rate selection on the generational time scale and multiple regression also can be used explain selection over arbitrarily scaled time intervals. While some have expressed strong opinions that the unit-time perspective is the more correct viewpoint (e.g., Caswell 2001:295) others acknowledge that selection acts directly also on the generation-scale (Lande 1982, Charlesworth 1994). For the purpose of measuring individual fitness, however, v_0 has a clear advantage: individual values of r are difficult to justify conceptually and their use in regressions lead to biased estimates of selection gradients (Lenski and Service 1982, McGraw and Caswell 1996), whereas selection gradients based on v_0 do not. It should be noted that generation-scaled measures of selection are convertible to unit-time measures simply by dividing by generation time.

Implementing multiple regressions.—The fundamental relationship between relative fitness, phenotypes, and selection gradients is defined succinctly by Lande and Arnold (1983) as $\beta = \mathbf{P}^{-1}\mathbf{s}$, where \mathbf{P} is the phenotypic variance-covariance matrix and \mathbf{s} is a vector of selection differentials. Each element s_i is the simple (as opposed to the partial) covariance between relative fitness and the trait z_i . A requirement for deriving the selection gradients is that the variance-covariance matrix \mathbf{P} be invertible. This condition is guaranteed when the matrix reflects all phenotype values for all individuals. When this condition is not met, then matrix singularities may cause multiple regressions to fail (Lynch and Arnold 1988). This is not a trivial problem, especially when observations of age-specific values of traits such as survival and reproduction depend upon earlier survival. In these cases, trait values are logically precluded from existing.

Nonexisting trait values are fundamentally different from missing values, as the latter term is most appropriately applied to traits that existed but were not observed (Haitovsky 1968, Allison 2001). One strategy to accommodate analyses with nonexisting trait values is to assume some particular distribution of the

unavailable data and/or to impute these unknowable values from some measure of the individuals' family or population (e.g., Lynch and Arnold 1988, Hadfield 2008, Shaw et al. 2008). However, it should be understood that implementing these approaches may add new phenotypic information to the multiple regression where none actually exists. As a result, descriptions of phenotypic selection may be unreliable. To insure against artificial fitness-phenotypic covariances, Moorad and Wade (2013) recommended for cases of nonexisting trait values (but not missing data) that the imputed values be taken from the average value of those individuals that expressed the trait. As long as an indicator variable is included as a trait in the multivariate analysis, then only the relevant multivariate information available to natural selection is included in the multiple regression. These indicator variables can be cumulative survival to the age of trait expression or a vector of age-specific survival rates p_x that multiply to give cumulative survival. This imputation strategy yields an invertible \mathbf{P} matrix suitable for deriving selection gradients. Importantly, the selection gradients are insensitive to the proportion of the imputed population ($1 - l_x$ in the present case), but the variance is discounted proportionally.

Recently, I applied this method to measure selection for female vital rates in a human population (Moorad 2013a). For this application, binary survival values at all ages up to the age of death are sufficient to serve as indicator variables. Individual age-specific survival and fertility rates at ages beyond death are imputed from the means of the surviving population; these means are the same as those conditional values that are employed in sensitivity analyses (i.e., those non-zero elements in Leslie matrices [Leslie 1945]). For any individual that dies at age d , the vital rates that contribute to \mathbf{P} are

$$p_{xi} = \begin{cases} 1 & x < d \\ 0 & x = d \\ \bar{p}_x & x > d \end{cases}$$

and

$$m_{xi} = \begin{cases} m_{xi} & x = d \\ \bar{m}_x & x > d \end{cases}. \quad (3)$$

The \mathbf{P} matrix implies variances that are attributed to the whole-population values; these are the variances of the vital rate that follows from the surviving fraction of the population, weighted by that fraction l_x . These weightings appear in the proof in Eqs. A.4, A.5, and A.8 in the Appendix and are grouped with other terms to produce gradients that are recognizable as Hamilton's sensitivities, but recall that selection is a covariance, and the discounting of this covariance that is caused by death ($1 - l_x$) is just as reasonably grouped with the variance term. The imputation strategy described in Eq. 3 will generate a \mathbf{P} matrix that reflects these discounts, which means that the selection gradient β that is solved

by the Lande-Arnold relationship will not. Differences between these selection gradients and those obtained by implicit differentiation and matrix manipulation are reconciled simply by multiplying the former by l_x .

APPLICATIONS

The supplied proof demonstrates that multiple regressions of v_0 upon all relevant vital rates must always yields the same selection gradients, scaled to generation time, for these vital rates as other methods that do not explicitly define individual fitness. An obvious interpretation of this result is that if population growth r measures mean population fitness over one time interval, then individual v_0 must define individual relative fitness over an entire generation. If so, then the definition of v_0 (Eq. 1) reveals that relative fitness is a linear function of vital rates \mathbf{z} ; the linear model of fitness is therefore completely described by

$$w(\mathbf{z}) = \boldsymbol{\beta}^T \mathbf{z} \quad (4)$$

where \mathbf{z} is a vector of vital rates up to, and including, the last age of reproduction in the population, and $\boldsymbol{\beta}$ are Hamilton's sensitivities. This simple relationship has profound implications for the study of the evolution of life histories, especially with regard to the measurement of natural selection. In the following sections, I discuss several useful applications of this perspective. All of these follow from conventional applications of multivariate regressions to understanding phenotypic selection, but all require explicit age-structured definitions of individual fitness that are neglected by sensitivity analyses.

Selection for other traits.—First, it is worth discussing a critical feature of the fitness function given by Eq. 4: there is no error term. All relative fitness variance is explained by first-order differences in vital rates at ages up to that age of the last reproductive event. In other words, directional selection for vital rates collectively explains the totality of the opportunity for selection. Consequently, when this full complement of vital rates is included in a model of phenotypic selection, there can be no selection for any other trait. This is consistent with Lande's perspective that the complete set of vital rates comprises a collection of characters that forms a necessary intermediate between fitness and other traits (Lande 1982). However, Lande's perspective differs slightly; his implies that quantifying phenotypic selection for some trait must be a two-step process that requires measuring (1) the association between some trait and all vital rates and (2) the association between all vital rates and fitness (quantified in terms of population growth rates). In contrast, the approach taken here allows phenotypic selection to be quantified by measuring directly the association between traits and v_0 . Accordingly, meaningful measurements of selection gradients for phenotypes that are not vital rates can be made, provided that *all* relevant vital rates are not included as covariates. For example, I recently measured

selection for adult mate number in a human population using Eq. 1 as a definition of fitness, but I used only cumulative survival to 15 years of age as a vital rate covariate (Moorad 2013b). This model of fitness allows for selection to act on cumulative mate number, but it is understood that it does so only through associations with survival and reproduction at ages greater than 15.

Expanding the linear model of fitness to include sex.—There are situations in which increasing the number of dependent variables is appropriate, but these will necessarily involve modifying the vital rate correlates. One highly useful application would be to measure sex-specific selection for vital rates. In this application, the linear model for fitness is

$$w_i = \beta_{wS} S_i + \boldsymbol{\beta}_M^T \mathbf{z}_M + \boldsymbol{\beta}_F^T \mathbf{z}_F \quad (5)$$

where S is the sex of the individual, and the subscripts M and F refer to sex-specific vital rates. $\boldsymbol{\beta}_M$ and $\boldsymbol{\beta}_F$ are the selection differentials for male and female vital rates, respectively. Selection for one sex or the other is β_{wS} , depending upon how the sexes are coded. For example, if males are 1 and females are 0, then there is selection against males when $\beta_{wS} < 0$ (this is expected when sex-ratios are male biased at birth, such as in human populations). Sex-specific vital rates that are not expressed in individuals of the other sex have values imputed from the means of surviving members of the opposite sex. Dependent variable values in the multiple regression (relative fitness) are found using Eq. 1, but after weighting age-specific reproduction by the inverse of the number of parents per child (i.e., 0.5 for two-parent reproduction and 1 for one-parent reproduction). Note that because the sex-specific linear model given in Eq. 5 contains all of the same information as the sex-independent model in Eq. 4, there is no error term, and all of the variance in relative fitness is explained by the set of independent variables.

Nonlinear selection.—Given that all relevant vital rates are included in the linear model (Eq. 4), all selection on vital rates is necessarily directional (or linear), and all fitness variance is explained by the variances and covariance of first-order vital rates \mathbf{z} . However, population perturbation methods have been applied to argue for ubiquitous nonlinear selection for vital rates (Caswell 1996, 2001, 2008). Resolving this issue is critical to understanding the evolution of vital rates because the existence of nonlinear vital rate selection implies that Hamilton's sensitivities present an incomplete description of vital rate selection. Recall that sensitivities ($\partial r / \partial z$), which can be viewed as first-order differentials of population growth rate taken with respect to vital rates, define directional selection gradients for vital rates (Hamilton 1966, Caswell 1978, Charlesworth 1994). The argument for nonlinear vital rate selection extends this interpretation to second-order differentials ($\partial^2 r / \partial z_x \partial z_y$), which are claimed to be equivalent to the quadratic terms defined by multiple regression (Caswell 1996, 2001, 2008).

Unfortunately, the theoretical basis for this interpretation is problematic because it is based upon phenotypic selection theory that treats individual fitness as a response variable (Lande and Arnold 1983, Phillips and Arnold 1989), but population growth is a group-level measure for which the approach was never intended. It is not self-evident that because first-order sensitivities successfully define selection gradients, it must follow that second-order sensitivities define stabilizing/disruptive selection (Lande and Arnold 1983, Phillips and Arnold 1989) or interaction selection (Brodie et al. 1995). For example, phenotypic selection theory makes clear that second-order fitness differentials can be expected to be equivalent to nonlinear selection gradients only when phenotypes are multivariate normal distributed (MVN). When this condition is not met, then estimates of nonlinear selection gradients must be made by multiple regressions of individual fitness on all first and higher-order terms simultaneously (this is not possible with sensitivity analyses because they lack the requisite covariances). With respect to vital rates, age-specific survival is binary, and fertility can be nearly so when age intervals are small relative to generation times (such as with annual reproduction in humans). Otherwise, age-specific fertility tends to be right-skewed. Clearly, the distributions of individuals' vital rates are far from MVN, and any higher order differentials of fitness taken with respect to vital rates cannot be relied upon to deliver valid estimates of nonlinear selection.

In fact, interpreting second order sensitivities as quadratic selection gradients can lead to results that are demonstrably incorrect. Let us consider a population with a simple three-age life history summarized by vital rates $\mathbf{p} = \{0.5 \ 0.5\}$ and $\mathbf{m} = \{0 \ 1 \ 2\}$. Using a population projection matrix constructed from these vital rates, I estimated the corresponding second-order sensitivities using the demogR package in R 2.9.0 (Jones 2007, R Development Core Team 2011). All second-order sensitivities were non-zero, including negative second-order sensitivities for fertility at age one and age two (-0.016 and -0.024 , respectively). Thus, the nonlinear selection interpretation identifies stabilizing selection for age-specific fertility at both ages. However, these specific vital rate means were chosen for this exercise because they lead to no population growth ($\lambda = 1$); it is well understood that relative fitness under these conditions is equal to the number of offspring (Caswell 2001:295, Charlesworth and Charlesworth 2010:505). When fitness added over some interval is directly proportional to the number of offspring produced, then, by definition, there can be no nonlinear relationship between fitness and age-specific fertility.

The opportunity for selection.—Selection gradients and differentials are popular comparative measures for studying natural selection for phenotypes (Kingsolver et al. 2001). Another metric for the strength of selection is the opportunity for selection, often identified by I , defined as the variation in relative fitness among

individuals in a population (or the mean-standardized variance [Crow 1958, O'Donald 1970, Wade 1979, Wade and Arnold 1980]). This variance is measured directly, and it cannot be reconstructed from life history tables. I is perhaps the most profound single measure of selection as it defines both the adaptive capacity of the population (Fisher 1958) and an upper boundary to the rate of evolution for specific traits (Hersch and Phillips 2004). Multivariate modifications of this concept have been applied to estimate relative fitness variance generated by specific episodes of selection (Crow 1958, Arnold and Wade 1984), and these represent the first steps in generalizing the opportunity for selection to age structure populations. In principle, these measures quantify the contribution of specific traits or life history stages toward the total adaptive potential of the population. However, the utility of these applications are made uncertain because episode-specific contributions toward I are correlated. Furthermore, the relationship between selection gradients and I were, until recently, unknown. These issues made it difficult to interpret components of I as finer scaled comparative measures of phenotypic selection (Arnold and Wade 1984).

However, Moorad and Wade (2013) recently derived I in terms of selection gradients, thereby unifying both systems of comparative phenotypic selection measures. The main result of this derivation is given in algebraic terms as

$$\mathbf{i}_z = \boldsymbol{\beta}_{wz} \mathbf{b}_{wz} \sigma_z^2 \quad (6)$$

where \mathbf{i}_z is a vector of additive, trait specific contributions to the relative fitness variance. Simply stated, this relationship identifies the direct contributing of a trait toward I as the product of the selection gradient acting on the trait (the partial regression coefficient of relative fitness on trait values, $\boldsymbol{\beta}_{wz}$) and the trait's selection differential ($\mathbf{b}_{wz} \sigma_z^2$, or the product of the simple regression of relative fitness on trait values and the trait variance). While the selection gradient tells us how selection will favor the change in a given trait, the component of I , i_z , quantifies the potential of specific trait variance to improve the fitness of the population by natural selection.

Recently, I applied the relationship given in Eq. 4 to the full set of vital rates in a human population (Moorad 2013a) with the primary goal of understanding how specific vital rates contribute independently to the adaptive potential of an age-structured population. This application improves upon Arnold and Wade's method of partitioning I into what they call "episodes of selection" (Arnold and Wade 1984) by providing non-overlapping components of fitness variance that are integrated into an evolutionary demographic perspective of selection that accounts for population growth. While there were some cohort-specific effects upon the relative and absolute contributions of vital rates to I in the analyzed human population, persistent and interesting

patterns emerged. First and foremost, survival variance at all ages collectively explained much less of the variance in fitness than did variation in reproduction (one-third to one-seventh of I was caused by survival variance, and the rest was caused by reproductive variance). In this population, therefore, there was two to six times more potential for fitness increase through fertility selection than through mortality selection. By focusing upon specific vital rates, I observed that the majority of the secular decline in the contribution of survival to fitness variance was caused by large decreases in first-year mortality. Shifts in reproductive behaviors caused large increases in fitness variance generated by early-age reproduction, but little change was generated by late-age reproduction.

Opportunity for selection components may offer an alternative to elasticities, or the associations between population growth rates and multiplicative changes in vital rates (Caswell 2001), as a means with which to compare selection for survival and fertility. Selection gradients are scaled by measurement units, such as number of children, or percent survived. In contrast, opportunities for selection components can be expressed as dimensionless quantities. This latter feature is viewed as a desirable property of elasticities. Because age-specific survival and fertility are scaled differently, elasticities are used by some researchers to compare the strength of selection among these two types of vital rates, but this practice is problematic as elasticities have no clear connection to the statistical relationships that define phenotypic selection (Caswell 2001:295). This is not a concern for opportunities for selection components, as they have clear context in the statistics of natural selection.

Contextual analysis.—Social interactions can shape the strength of selection for phenotypes. Multilevel selection and inclusive fitness are alternative approaches for quantifying these forces of selection. While there is a history of contentious debate about which approach is better, there is general agreement that they measure the same processes (see Okasha [2006] for an overview on this subject). Contextual analysis is a multivariate regression-based approach used to measure multilevel selection by discriminating between sources of “individual-level” and “group-level” selection (Heisler and Damuth 1987, Damuth and Heisler 1988, Goodnight et al. 1992, Okasha 2004). Here, individual-level selection gradients are the partial regression coefficients that describe the association between individual fitness and the individual phenotypes, holding the attributes of the group constant. These are the context-free components of natural selection (or simply individual-level selection). Group-level selection is defined by the partial regression coefficient of fitness on the phenotypes of the group, holding the phenotypes of the individuals constant. These are the contextual components of selection. Alternatively, group-selection can be said to describe the association between fitness and emergent

properties of groups, including social interactions. Multilevel selection for some trait z is can be defined as

$$w_i = \mu + \beta_{w,z}z + \beta_{w,z'}z' + \varepsilon_i \quad (7)$$

where z is the context-free individual phenotype measure, usually taken as the difference between the individual's phenotype and the mean of its social partners, and z' is the mean of the social partners' trait values.

As contextual analysis can be extended to multivariate phenotypes, it is correctly viewed as the Lande-Arnold phenotypic selection definition (Lande and Arnold 1983) generalized to include the effects of social interactions (Heisler and Damuth 1987). Accordingly, contextual analysis takes the perspective that fitness is an attribute of the individual (not the group), although it allows for traits that are attributes of the individual (z) or the group context of the individual (z'). This critical identification of individual fitness means that contextual analysis and sensitivity analysis are not compatible. However, the definition of individual fitness supplied in Eq. 1 provides a simple method for obtaining the necessary dependent variables to apply to a contextual analysis of multilevel selection in age-structure populations. I recently applied this strategy to measure family-level selection for mating success in a human population (Moorad 2013b), and I found that individual-level selection and family-level selection (a form of group-level selection), operating through both paternal and maternal effects, favored increased mate numbers in both sexes. As mentioned previously, this analysis used a vital rate (cumulative survival to age 15) as a covariate. It should be noted, however, that if all vital rates are included in the linear model, then the analysis will yield no group-level selection as variation in individual vital rates must explain all fitness variance. One can interpret this to mean that group-level selection can never act directly on vital rates.

However, there need be no such constraints operating on the relationship between vital rates and other traits, and contextual analysis applied to specific vital rates instead of relative fitness can be used to resolve how specific vital rates contribute to multilevel selection acting on some trait of interest. For some individual trait z and contextual trait z' , individual-level selection in an age-structured population is

$$\text{cov}(v_0, z) = \text{var}(z) \sum_x (\beta_{wP_x} \beta_{P_x z} + \beta_{wm_x} \beta_{m_x z}) \quad (8a)$$

and group-level selection is

$$\text{cov}(v_0, z') = \text{var}(z') \sum_x (\beta_{wP_x} \beta_{P_x z'} + \beta_{wm_x} \beta_{m_x z'}) \quad (8b)$$

where β_{wP_x} and β_{wm_x} are the selection differentials for vital rates defined by Hamilton's sensitivities. The partial regression coefficients $\beta_{P_x z}$ and $\beta_{P_x z'}$ follow from a multiple regression of age-specific survival on individual and group attributes of the trait of interest (other

regressions are used to determine $\beta_{m,z}$ and $\beta_{m,z'}$. Because these expressions trace the generation of selection through each vital rate independently, they can be considered to be a generation-scaled version of Lande's age-structured phenotypic selection model (Lande 1982) generalized to multilevel selection.

Limits to the phenotypic selection perspective.—A phenotypic response to selection requires phenotypic selection (how fitness differentially weights the phenotypes that are chosen to contribute to the next generation) and phenotypic transmission (the degree to which parental phenotypes are faithfully inherited by their offspring). The purpose of this paper is to comment on the nature of the former processes, but it is entirely unconcerned with the nature of the latter, which is the domain of quantitative genetic concepts such as heritability, genetic correlations, and ultimately gene frequencies. It is important to recognize that these genetic relationships can be quite complex, and it does not necessarily follow that quantitative, or even qualitative, assessments of selection gradients accurately reflect a multivariate response to selection. Consider, for example, that Hamilton's model of aging (Hamilton 1966) assumes a particular relationship between vital rates and gene frequencies to arrive at one predicted evolutionary endpoint (Charlesworth 1994). However, different models of vital rate genetics can lead to very different endpoints (e.g., Charlesworth 2001, Baudisch 2005, Moorad and Promislow 2008, 2011, Wachter et al. 2013) despite retaining Hamilton's essential map of fitness on vital rates.

While the goals of a phenotypic selection study are more modest in scope than a study of phenotypic evolution, descriptions of phenotypic selection are freed from some assumptions and caveats that are often necessary for tractable genetic solutions. For example, population genetic models of age-structured populations emphasize that rates of gene frequency change are only approximately proportional to variation in the fitness of genotypes (as quantified by growth rates, r), and evolutionary predictions are often qualified as approximately valid with weak selection only (Norton 1928, Charlesworth 1994), but there is no clear reason to expect that this condition applies to the present discussion of phenotypic selection where population growth rates are empirically determined. However, the phenotypic selection methods outlined here do assume that a demographic equilibrium holds with respect to population growth rates and age-structure. As this condition is not likely to be met in most real populations, resulting estimates of selection are best viewed as approximations.

CONCLUSION

While sensitivity analysis can be a useful tool for estimating selection in age structured populations, it is neither as flexible nor as powerful as multivariate regression. Individual measures of fitness are required

for the latter, however, and a few different definitions have been proposed for situations where populations are age structured (e.g., McGraw and Caswell 1996, Peters and Keightley 2000, Coulson et al. 2006). According to Fisher, reproductive value quantifies "To what extent will persons of this age, on the average, contribute to the ancestry of future generations?" Applying this concept to individuals rather than to groups of individuals of the same age suggests a sensible verbal definition of individual fitness in age structured populations. Reproductive value at birth (v_0) is the only proposed definition of individual fitness that has been shown to (1) converge with total reproductive output when population size does not change, (2) lack statistical bias when applied to individuals, and (3) provide selection gradients that agree with Hamilton's sensitivities. It is reasonable to view these as necessary conditions for a valid definition of individual fitness.

Fisher had famously asserted that the strength of selection that acts at some age should be proportional to the reproductive value at that age (Fisher 1958). This view has long been considered fallacious (Hamilton 1966, Rose 1991, Charlesworth 1994, 2000), but it should be understood that Fisher's use of reproductive value applies to the mean of individuals of the same age. As I showed here, v_0 for individuals provides selection measurements that are wholly compatible with the correct predictions, as derived by Hamilton (1966). Evidently, few have used v_0 as the lifetime fitness of individuals (Peters and Keightley 2000, Moorad 2013a, b), but others have used v_0 averaged over individuals with common genotypes to define the fitness of genotypes (Charlesworth and Charlesworth 1973, Charlesworth 1994, Tatar and Promislow 1997).

I have discussed how multivariate regression equipped with an appropriate definition of individual fitness can be used to measure phenotypic selection for traits relevant to life history evolution. I focused on situations where sensitivity analysis cannot be applied (opportunity for selection and contextual analysis) or where it has led to misleading inferences (nonlinear selection). Given that the assumptions that must hold are the same for the two strategies (constant growth rate and stable age structure), there appears to be no reason to prefer sensitivity analyses to multivariate regression when complete individual data are available. When only population-level data are available (such as might be summarized in a published life table, for example), then sensitivity analyses can be performed when regressions cannot. Otherwise, multivariate regressions offer a simple, flexible, and powerful approach to understanding phenotypic selection in age-structured populations.

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SUPPLEMENTAL MATERIAL

Appendix

Proof that regressing individuals' reproductive values on vital rates yields Hamilton's sensitivities ([Ecological Archives E095-092-A1](#)).